THE DECOMPRESSION SICKNESS AND VENOUS GAS EMBOLI CONSEQUENCES OF AIR BREAKS DURING 100% OXYGEN PREBREATHE

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INTRODUCTION: Not enough is known about the increased risk of hypobaric decompression sickness (DCS) and production of venous (VGE) and arterial (AGE) gas emboli following an air break in an otherwise normal 100% resting oxygen (O₂) prebreathe (PB), and certainly a break in PB when exercise is used to accelerate nitrogen (N₂) elimination from the tissues. Current Aeromedical Flight Rules at the Johnson Space Center about additional PB payback times are untested, possibly too conservative, and therefore not optimized for operational use. **HYPOTHESIS**: A 10 min air break at 90 min into a 120 min PB that includes initial dual-cycle ergometry for 10 min will show a measurable increase in the risk of DCS and VGE after ascent to 4.3 psia compared to a 10 min break at 15 min into the PB, or when there is no break in PB. METHODS: Data collection with humans begins in 2005, but here we first evaluate the hypothesis using three models of tissue N₂ kinetics: Model I is a simple single half-time compartment exponential model, Model II is a three compartment half-time exponential model, and Model III is a variable half-time compartment model where the percentage of maximum O₂ consumption for the subject during dual-cycle ergometry exercise defines the half-time compartment. RESULTS: Model I with large rate constants to simulate an exercise effect always showed a late break in PB had the greatest consequence. Model II showed an early break had the greatest consequence. Model III showed there was no difference between early or late break in exercise PB. CONCLUSIONS: Only one of these outcomes will be observed when humans are tested. Our results will favor one of these models, and so advance our understanding of tissue N₂ kinetics, and of altitude DCS after an air break in PB.